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1: Blood 1994 Oct 1;84(7):2182-8 Related Articles, Links

Human macrophage colony-stimulating factor is expressed at and shed from the cell surface.

Tuck DP, Cerretti DP, Hand A, Guha A, Sorba S, Dainiak N.

Department of Medicine, Royal Victoria Hospital, McGill University, Montreal, Canada.

Surface membrane-associated growth factors are being recognized as important for developmental processes, including cell assembly, differentiation, and growth. To investigate the role of membrane-bound macrophage colony-stimulating factor (M-CSF) in myelopoiesis, and whether this factor is released from the cell surface in association with shed membrane-derived vesicles, COS-1 cells were transfected with cDNAs for M-CSF-tau (containing the transmembrane domain) or a soluble mutant form of the molecule lacking the transmembrane domain ([s]M-CSF-alpha). COS-1 cells transfected with either cDNA released activity into the spent culture medium. Conditioned medium was separated by centrifugation into supernatants and pellets were found to contain plasma membrane-derived vesicles by transmission electron microscopy. When medium fractions were assayed in marrow cultures, activity was localized to shed plasma membrane-derived vesicles in medium conditioned by cells transfected with cDNA for M-CSF-tau and in the vesicle-free supernatants of medium conditioned by cells transfected with cDNA for [s]M-CSF-alpha. In addition, nuclear, mitochondrial, and plasma membrane subfractions of stably transfected cells were prepared and assayed for activity. Concentration-dependent stimulation of macrophage colony formation was observed with purified plasma membranes (but not nuclear or cytosolic proteins) from cells transfected with cDNA for M-CSF-tau. By contrast, membranes from untransfected cells and cells transfected with cDNA for [s] M-CSF-alpha or control DNA expressed no activity. Together, the data indicate that human M-CSF is expressed at the cell surface and exfoliated in association with surface membrane-derived vesicles.

PMID: 7919334 [PubMed - indexed for MEDLINE]

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1: Exp Cell Res 1994 Jan;210(1):107-12

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A glycosylphosphatidylinositol-anchored cytokine can function as an artificial cellular adhesin.

Weber MC, Groger RK, Tykocinski ML.

Institute of Pathology, Case Western Reserve University, Cleveland, Ohio 44106.

A novel strategy for altering the adhesive properties of cells has been developed which is based upon the use of artificial adhesins. Specifically, a glycosylphosphatidylinositol (GPI)-modified variant of the cytokine macrophage colony stimulating factor (M-CSF), designated M-CSF.GPI, was expressed on the surface of human bone marrow stromal cells. A chimeric M-CSF:decay-accelerating factor expression construct was used for M-CSF.GPI expression. Cell:cell binding assays established that this artificially membrane-tethered cytokine functions as a potent cellular adhesin, allowing for enhanced binding to M-CSF receptor-expressing cellular transfectants. Antibody blocking analyses confirmed the M-CSF:M-CSF receptor dependence of the enhanced intercellular binding. This capacity to direct the cellular interactive repertoire of selected cells can in principle be applied to other cell types and other molecular pairs to be used in cell-based therapies.

PMID: 8269987 [PubMed - indexed for MEDLINE]

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☐ 1: Blood 1996 Jun 15;87(12):5232-41

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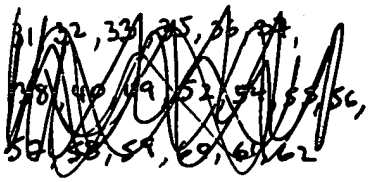
Macrophages can recognize and kill tumor cells bearing the membrane isoform of macrophage colony-stimulating factor.

Jadus MR, Irwin MC, Irwin MR, Horansky RD, Sekhon S, Pepper KA, Kohn DB, Wepsic HT.

Department of Laboratory Service, Veterans Affairs Medical Center, Long Beach, CA 90822, USA.

NBXFO hybridoma cells produced both the membrane and secreted isoforms of macrophage colony-stimulating factor (M-CSF). Murine bone marrow cells stimulated by the secreted form of M-CSF (sM-CSF) became Mac1+, Mac2+, Mac3+, and F4/80+ macrophages that inhibited the growth of NBXFO cells, but not L1210 or P815 tumor cells. In cytotoxicity studies, M-CSF activated macrophages and freshly isolated macrophages killed NBXFO cells in the presence of polymyxin B, eliminating the possibility that contaminating lipopolysaccharide (LPS) was responsible for the delivery of the cytotoxic signal. Retroviral-mediated transfection of T9 glioma cells with the gene for the membrane isoform of M-CSF (mM-CSF), but not for the secreted isoform of M-CSF, transferred the ability of macrophages to kill these transfected T9 cells in a mM-CSF dose-dependent manner. Macrophage-mediated killing of the mM-CSF transfected clone was blocked by using a 100-fold excess of recombinant M-CSF. Catalase, superoxide dismutase, and the nitric oxide inhibitor, N-omega-nitro-arginine methyl ester (NAME), did not effect macrophage cytotoxicity against the mM-CSF transfectant T9 clones. T9 parental cells when cultured in the presence of an equal number of the mM-CSF transfectant cells were not killed, indicating specific target cell cytotoxicity by the macrophages. Electron microscopy showed that macrophages were capable of phagocytosizing mM-CSF bearing T9 tumor cells and NBXFO hybridoma cells; this suggested a possible mechanism of this cytotoxicity. This study indicates that mM-CSF provides the necessary binding and triggering molecules through which macrophages can initiate direct tumor cell cytotoxicity.



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PMID: 8652838 [PubMed - indexed for MEDLINE]

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L1210 cells are mouse cells that are derived from tumor

1: Exp Hematol 1996 Feb;24(2):360-3 Related Articles, Links

Augmentation of antitumor immunity using genetically M-CSF-expressing L1210 cells.

Kimura F, Douzono M, Ohta J, Morita T, Ikeda K, Nakamura Y, Sato K, Yamada M, Nagata N, Motoyoshi K.

The Third Department of Internal Medicine, National Defense Medical College, Saitama, Japan.

Macrophage colony-stimulating factor (M-CSF) enhances tumoricidal activities of macrophages. We transduced human M-CSF cDNA into the mouse lymphoid cell line, L1210, and examined the antitumor effect of the locally expressed M-CSF. Mice injected with the M-CSF-producing subline showed improved survival in comparison with the mock-transfected cell line or parental cell line plus M-CSF administration (20 microg/kg for 3 days) at inoculated cell numbers of 10(2) or 5 x 10(3). The survival rate at 50 days after injection of 10(6) high M-CSF-expressing cells was 80%, significantly higher than that after injection of the mock-transfected cells, which killed all the mice by day 23. The survival rate appeared to depend on the amount of M-CSF produced. Moreover, all surviving mice after intravenous injection of the M-CSF-expressing sublines were rechallenged with 10(6) parental L1210 cells at day 50, and all survived up to day 100, demonstrating that M-CSF-expressing cells induced immune protection against the parental cells. The same improvement of survival was observed in mouse M-CSF-expressing cell lines. These observations imply that M-CSF cDNA is a candidate gene for use in gene therapy in leukemia.

PMID: 8641366 [PubMed - indexed for MEDLINE]

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